deliveries of suitable bread and biscuits can be arranged. (Welfare Foods (Stockport) Ltd., Swallow Street, Stockport, Cheshire.)—I am, etc.,

The Children's Hospital,
Sheffield 10.

K. S. HOLT.

REFERENCES

- ¹ Holt, K. S., *Acta paediat.*, 1963, **52**, 417.
- ² Blainey, J. D., and Gulliford, R., *Arch. Dis. Child.*, 1956, **31**, 452.
- ³ Allan, J. D., and Moss, A. D., in *Neurometabolic Disorders in Childhood*. Ed. K. S. Holt and J. Milner, 1964. Livingstone, Edinburgh.

Waldenström's Macroglobulinaemia

SIR,—The pace of scientific progress is now so great that "facts" tend to be disproved before they appear in print. In your leader (9 January, p. 73) you state that: "All three [immunoglobulins] are composed basically of two pairs of polypeptide chains." Franklin's¹ latest chemical studies (published in November 1964) suggest, however, that the "H chain" is not a single polypeptide as had been commonly assumed, but that it consists of two chains linked by an unusual bond. These latest observations support Comings's² model of immunoglobulin structure, which was based upon an analysis³ of genetic and autoimmune⁴ evidence. Cohen and Porter⁵ cite additional biological evidence in support of a 6-chain model. It is possible that classical (L-chain) Bence-Jones proteins which reveal homogeneous and heterogeneous peptide fragments may also prove to be complexes where the homogeneous portion corresponds to classical type I or II L chains, and the heterogeneous portion corresponds to Fd chains,⁶ which—according to the autoimmune evidence—should be encoded by X-linked genes.^{3, 6} The Fd chain is of the utmost biological importance because it contains the antibody-combining site, and it should therefore largely determine its specificity.¹ Complete agreement between the theoretical deductions from the autoimmune evidence and direct chemical observation of the basic structure of IgG globulins would appear to be imminent.—I am, etc.,

The General Infirmary,
Leeds 1.

P. R. J. BURCH.

REFERENCES

- ¹ Franklin, E. C., *J. exp. Med.*, 1964, **120**, 691.
- ² Comings, D. E., *Lancet*, 1963, **2**, 786.
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- ⁴ Lawrence, J. S., *Ann. rheum. Dis.*, 1961, **20**, 11.
- ⁵ Cohen, S., and Porter, R. R., *Advanc. Immunol.*, 1964, **4**, 287.
- ⁶ Burch, P. R. J., Burwell, R. G., and Rowell, N. R., *Lancet*, 1964, **1**, 720.

Renal Hypertension and Polycythaemia

SIR,—On the basis of the interesting case which they describe, Dr. R. G. Luke and his colleagues (16 January, p. 164) suggest that where polycythaemia and hypertension occur together renal-artery stenosis should be considered, and this suggestion is further advocated in your leading article (16 January, p. 138). While accepting this recommendation, we wish to point out that in the presence of hypertension it is more desirable than usual that the diagnosis of polycythaemia should rest on measurements of total red-cell volume, as in Dr. Luke's patient, rather than on venous haematocrit and haemoglobin levels only. In a group of patients with uncomplicated essential hypertension it was found that

neither red-cell volume nor plasma volume showed a systematic deviation from normal values, but that venous haematocrit and whole-body haematocrit varied over a wider range than normal.¹ Moreover, we have studied three patients with essential hypertension in whom the venous haematocrit was 57%, equal to that of Dr. Luke's patient, but in whom the red-cell volumes were not elevated (25.3, 26.6, and 30.2 ml./kg.).

It is also noteworthy that in a patient with polycythaemia, hypertension, and unilateral hydronephrosis (Case 1 of Jones *et al.*)² nephrectomy was followed by disappearance of the polycythaemia but not of the hypertension. This result exemplifies the difficulties of forecasting the effects of nephrectomy in such a patient.

Hypertension and polycythaemia may also simultaneously complicate phaeochromocytoma.³ Both complications disappeared after removal of four phaeochromocytomas from the patient described by the latter authors. In addition to renal-artery stenosis a phaeochromocytoma should therefore be considered when polycythaemia and hypertension co-exist, and in either case the diagnosis of polycythaemia should depend preferably on measurement of red-cell volume.—We are, etc.,

N. F. JONES.

M. A. BARRACLOUGH.

W. F. CLAPHAM.

St. Thomas's Hospital,
London S.E.1.

REFERENCES

- ¹ Jones, N. F., Clapham, W. F., Barracough, M. A., and Mills, I. H., *Clin. Sci.*, 1964, **26**, 307.
- ² ———, Payne, R. W., Hyde, R. D., and Price, T. M. L., *Lancet*, 1960, **1**, 299.
- ³ Waldmann, T. A., and Bradley, J. E., *Proc. Soc. Exp. Biol. (N.Y.)*, 1961, **108**, 425.

Gas-cylinder Keys

SIR,—The group engineer's department has made for me a key which avoids the difficulties set out by Dr. H. Lister Wilson (19 December, p. 1591). It consists simply of a T made from mild steel pipe 7 in. (17.5 cm.) long in its lower limb, the end of which is shaped to fit the cylinder.—I am, etc.,

Hastings, Sussex.

HARRY MIDDLETON.

Code for Drugs

SIR,—Dr. W. A. L. Collier (26 December, p. 1657) suggests that the stilboestrol tablets in the Co-Tabs range should have been marked "SJ 2 . . . SJ 7" instead of "S 3 . . . S 20." I am told that, for technical reasons, his suggested markings would not have been possible on the smaller stilboestrol tablets. Manufacturing considerations aside, the code used on Co-Tabs was chosen because it was simple. A coded tablet may have to be identified by a general practitioner late at night in a country cottage, or by a tired hospital casualty officer in the early hours of the morning; and the code may have been little studied by them beforehand. Most of the systems of tablet coding that have been suggested in the past do not satisfy this criterion of simplicity; and neither does any system yet devised for identifying existing uncoded tablets.

Dr. H. J. Cronhelm (9 January, p. 129) says that all we need for tablet identification is a universal code number to be applied to the prescription label at the time of dispensing. Similarly Dr. W. A. L. Collier (26 December, p. 1657) suggests that a system of abbreviations for drug names should be used in the marking of tablets. I think both suggestions have merit, but who would, in fact, devise the universal code numbers for the labels or the drug name abbreviations? Who would ensure their use? And above all, when might usage be expected to start? The Co-Tabs system exists, is in everyday use, is simple, and it works. What more is to be looked for?—I am, etc.,

S. BRADSHAW,

Medical Adviser,

Arthur H. Cox and Co., Ltd.

Amersham, Bucks.

Bacterial Agglutinating Antisera and Immunofluorescence

SIR,—A word of warning should be given to those who may contemplate using routine diagnostic agglutinating antisera for the identification of micro-organisms by immunofluorescence.

Immunofluorescence detects antigens, but it does not differentiate between organisms having antigens in common. The diagnostic antisera issued by the Standards Laboratory are not cross-tested against organisms which are routinely excluded by means of initial biochemical tests, particularly the use of selective media. The content of antibodies to such organisms is therefore not known, nor the extent of variation of these antibodies from one batch of antiserum to another. Because of these limitations some antisera may prove successful in a study of this kind whereas later batches of the same antisera may not.

We issue bacterial diagnostic antisera in the belief that they will be used only in established agglutination tests. The intelligent use of the antisera in any other way requires full knowledge of the method of preparation. We therefore hope that anyone trying out new techniques will, in their own interest, let us know details of the investigations contemplated before applications for antisera are made.—I am, etc.,

PATRICIA BRADSTREET.

The Standards Laboratory for Serological

Reagents,

Central Public Health Laboratory,

London N.W.9.

Cephaloridine in Chronic Bronchitis

SIR,—The manufacturers of cephaloridine (Ceporin) state in the literature supplied with this antibiotic that the mean inhibitory concentration (M.I.C.) for *Haemophilus influenzae* is 2.0–3.0 µg./ml. and that the maximum levels obtained in the blood with the dose recommended is of the order of 12 µg./ml. Barber and Waterworth¹ showed that the M.I.C. of 12 strains of *H. influenzae* was 4–16 µg./ml., and Muggleton and his colleagues² that it was 1–8 µg./ml. for three strains. Stewart and Holt³ found the M.I.C. to be 0.1–5 µg./ml. with capsulated strains.

We have tested 52 strains of *H. influenzae* recently isolated from patients with acute exacerbations of chronic bronchitis, none of